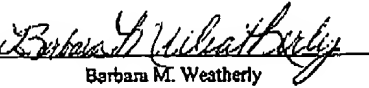


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Attorney Docket No.: 15270J-004740US
Client Ref. No. 209-US-CIP5

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JUN 27 2006

**IN THE UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES**

In re application of:

DALE B. SCHENK

Application No. 09/322,289

Filed: May 28, 1999

For: PREVENTION AND TREATMENT OF
AMYLOIDOGENIC DISEASE

Confirmation No. 7773

Examiner: Sharon L. Turner

Art Unit: 1649

**APPELLANT'S BRIEF
UNDER 37 C.F.R. § 41.37**

Mail Stop Appeal Brief - Patents
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

Further to the Notice of Appeal mailed February 27, 2006 in the above-referenced application, Appellant submits this Brief on Appeal along with the fee set forth under 37 C.F.R. § 41.37. Appellant submits a Petition for Extension of Time from April 27, 2007 to June 27, 2006 herewith.

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1. **REAL PARTY IN INTEREST**

Elan Pharma International Limited

2. **RELATED APPEALS AND INTERFERENCES**

An appeal is pending in related case US Application No. 09/723,765. However, the claims and issues are significantly different from those in the present case.

3. **STATUS OF CLAIMS**

Claims 1, 2, 4, 6-8, 10-12, 17, 21-24, 31, 32, 35-37, 82-90 and 93-102 stand rejected and are appealed. Claims 25-28, 33, 34, 38-58 and 60-81 are withdrawn. Claims 3, 5, 9, 13-16, 18-20, 29, 30, 59, and 91-92 are cancelled.

4. **STATUS OF AMENDMENTS**

No amendment after final has been filed.

5. **SUMMARY OF CLAIMED SUBJECT MATTER**

Claim 1 is directed to a method of treating a disease characterized by an amyloid deposit comprising A β peptide (specification at p. 10, lines 8-10). The method entails administering to a patient having the disease an antibody that specifically binds to A β peptide, in a regime effective to treat the disease (specification at p. 14, lines 14-16 and Example XI at p. 70 *et seq.*). The antibody is a chimeric or humanized antibody, or a human monoclonal antibody and the antibody is of isotype human IgG1 (specification at p. 21, lines 15-20). Independent claim 82 is directed to a method of prophylaxis of a disease characterized by an amyloid deposit comprising A β peptide (specification at paragraph bridging pp. 26-27). The method entails administering to a patient an antibody defined in the same manner as claim 1 in a regime effective to effect prophylaxis of the disease.

Dependent claims 22, 23, 95 and 96 specify dosages of wherein least 1 mg/kg and 10 mg/kg body weight are administered.

Dependent claims 31 and 98 specify an antibody that binds to A β without binding to amyloid precursor protein.

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6. GROUND OF REJECTION TO BE REVIEWED ON APPEAL

Whether claims 1-2, 4, 6-8, 10-12, 14-15, 17, 21-24, 31-32, 35-37 and 82-102 would have been obvious over Nettleship, EP 613,007 [Nettleship], Walker *et al.*, J. Neuropathol. Exp. Neurol., 53, 377-83 (1994) [Walker], Better *et al.*, US 5,576,184 [Better], and Friedland *et al.*, Mol. Neurobiol. 9, 107-113 (1994) [Friedland]

Claims 1-2, 4, 6-8, 10-12, 14-15, 17, 21-24, 32-32, 35-37 and 82-102 also stand rejected for obviousness-type double patenting over US 6,761,888 and US 6,743,427. However, appellant has agreed to file a terminal disclaimer if the claims are allowed in their current form. Therefore, the Board need not review the issue on the merits.

7. ARGUMENT

7.1 The Examiner's Rationale

The Examiner's rationale is provided at pp. 8-13 of the final office action of November 29, 2005. Nettleship is alleged to teach use of antibodies for treatment of Alzheimer's disease (final office action at p. 8, second paragraph). The Examiner concedes that Nettleship does not teach selection of the human IgG1 isotype of antibody (final office action at p. 10, second paragraph). Walker is alleged to teach in vivo labeling of cerebral amyloid using an IgG1 isotype antibody (final office action at p. 10, second paragraph). The Examiner further concedes that "Walker does not teach administration of the antibody for treatment purposes." Better is alleged to teach chimeric or humanized monoclonals of the IgG1 isotype to provide advantages of decreased hyperimmunogenicity as well as prolonged half-life (final office action at p. 11, second paragraph). Friedland is alleged to teach the proper dosage to a mammal such as a human (final office action at p. 12, first paragraph).

7.2 Summary of the References

7.2.1 Nettleship

Nettleship is an entirely prophetic application that discusses several assays to identify compounds that inhibit the neurotoxicity of A β cells in culture (columns 4 and 5), and speculates that compounds thereby identified might be useful for treating Alzheimer's disease (col. 1, lines 49-51). The reference indicates that A β in beta sheet form is toxic in such assays, whereas A β in random coil form is only minimally so. The reference further speculates that two classes of

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antibodies might be useful in therapeutic and/or diagnostic methods. One class is antibodies that specifically bind to A β in beta sheet form without binding to A β in random coil form (col. 5, lines 45-47). The other class has the opposite binding specificity, that is, antibodies that specifically bind A β in random coil form without binding to A β in beta sheet form (col. 5, lines 47-50). The reference also speculates that the former class might be incorporated in pharmaceutical compositions (see abstract and column 2, lines 5-10).

7.2.2 Walker

Walker reports that a mouse antibody to A β injected into the CNS of monkey brains can label some amyloid deposits within the brains. The 10D5 antibody used in the study was a mouse antibody having mouse IgG1 isotype. Walker also comments on several obstacles to be overcome before such labelling can be used for diagnostic purposes. First, the extent of labelling even from CNS injection was incomplete ("intracisternally injected antibody failed to reach a significant number of deposits") (at p. 382, second paragraph). Second, intracistinal injection or other techniques used to circumvent the blood brain barrier are "not suitable for routine therapeutic or diagnostic purposes" (at p. 382, second paragraph). Third, the "blood-brain barrier prevents the passage of many types of molecules from the bloodstream to the brain . . . rendering vascular delivery of ligands to A β problematic" (at p. 377, first column, first paragraph). Walker concludes only that it "may eventually be feasible to employ antibodies to deliver therapeutic agents directly to A β in the brain, or in combination with imaging technologies, such as PET or SPECT, to diagnose β -amyloidoses in living subjects" (at p. 381, first column, second paragraph, emphasis supplied).

7.2.3 Friedland

Friedland discusses using a mouse antibody to stain amyloid deposits in post-mortem brain sections from Alzheimer's patients with a view to developing an in vivo imaging agent. In Friedland's experiments the Fab form of the antibody stained similarly to the intact antibody (see Abstract). Friedland expresses a preference for Fab fragments over intact antibody because of their more rapid clearance (p. 112, paragraph bridging cols. 1 and 2). Like Walker, Friedland comments on the difficulty imposed by the blood brain barrier for detecting plaques in living subjects ("We do not anticipate passage of the labeled MAb through the blood-brain barrier [citations omitted] will cause MAb binding in Alzheimer's disease brain," p. 112, second column, first paragraph).

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7.2.4 Better

Better discusses production of chimeric antibodies and comments on the improved immunogenicity and half-life of chimeric antibodies relative to mouse antibodies. However, such improvements are attributed to the "human specific" properties of chimerics (Better at col. 3, lines 42-52) rather than to any particular human isotype.

7.3 Claims 1-2, 4, 6-8, 10-12, 14-15, 17, 21-24, 31-32, 35, 37 and 82-102 Would Not Have Been Obvious over Nettleship, Friedland, Walker and Better

7.3.1 The Office Action Lacks the Specificity Required to State a Prima Facie Case of Obviousness

In proceedings before the Patent and Trademark Office, the examiner bears the burden of establishing a prima facie case of obviousness based upon the prior art (*In re Piasecki*, 223 USPQ 785, 787-88 (Fed. Cir. 1984)). A statement of a rejection must explain with reasonable specificity the basis of the rejection; otherwise the examiner procedurally fails to establish a prima facie case of obviousness. *Ex parte Blanc*, 13 USPQ2d 1383 (Bd. Pat. App. & Intf. 1989). "Broad conclusory statements regarding the teaching of multiple references" are insufficient to establish obviousness, *In re Dembiczak*, 50 USPQ2d 1614, 1617 (Fed. Cir. 1999).

Here, the Examiner has cited four references, Nettleship, Walker, Better, and Friedland but has not identified with particularity which references are being applied in the alternative and which in combination, or if certain references are only cited with respect to certain dependent claims. For example, it is unclear whether Better is being applied in combination with Walker or in the alternative, or both of these. Further, the initial statement of rejection applies Friedland against all of the claims, but the Examiner's later remarks (final office action at p. 12, first paragraph) only refer to this reference in connection with dosage. The statement of rejection concludes by stating that the "claimed invention is rendered obvious in light of the cumulative reference teachings." (Final office action at p. 12, second paragraph). The conclusory reference to the cumulative teachings without delineation of how the references are being combined forces appellant to guess at the nature of the rejection to formulate a response as can be seen from the remarks below. In these circumstances, it is respectfully submitted that the rejection is stated with insufficient clarity to constitute a prima facie case of obviousness.

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7.3.2 No Combination of Nettleship, Walker, Better or Friedland
Rendered the Claimed Invention Obvious

The claimed invention specifies methods of treatment or prophylaxis of Alzheimer's disease using an antibody to A β having human IgG1 isotype. In accordance with convention, the isotype of the antibody characterizes its heavy chain constant region. The human IgG1 isotype is believed to be advantageous for use in treatment because it interacts most strongly with the human receptor Fc γ R1. Different nomenclature is used to describe human and mouse isotypes. Thus, the mouse equivalent of human IgG1 is mouse IgG2a, not mouse IgG1 (Paul, *Fundamental Immunology*, Raven Press (1993), p. 838.). Post filing evidence by the present inventor and colleagues confirms the advantageous selection of the human IgG1 isotype or its mouse equivalent Ig2a (see WO 00/72880, IDS at p. 18, lines 15-17 and Bard *et al.*, PNAS 100, 2023-2028 (2003) IDS, p. 2027, second column, third paragraph). However, before the priority date it was not obvious which human isotype would be advantageous for treatment or prophylaxis of Alzheimer's disease or for what reason.

The prior art references when combined must teach or suggest all of the claim limitations. *In re Vaeck*, 20 USPQ2d 1438 (Fed. Cir. 1991). "To establish a prima facie case of obviousness based on a combination of the content of various references, there must be some teaching, suggestion or motivation in the prior art to make the *specific* combination that was made by the applicant." *In re Dance*, 48 USPQ2d 1635, 1637 (Fed. Cir. 1998) (emphasis supplied). The motivation must have sufficient "force" to "impel persons skilled in the art to do what applicant has done." *Ex parte Levengood*, 28 USPQ2d 1300, 1302 (BPAI 1993). A reference teaching away from an invention is strong evidence of non-obviousness, in fact, the very antithesis of obviousness, to which a rebuttal should not even be required. *In re Buehler*, 185 USPQ 781 (CCPA 1975); *In re Hedges*, USPQ 685, 687 (Fed. Cir. 1986).

Here, the Examiner acknowledges that Nettleship does not teach use of an antibody to A β having human IgG1 isotype. In appellant's submission, none of Walker, Better and/or Friedland remedies this deficiency. The 10D5 antibody used by Walker is of mouse IgG1 isotype. Not only is mouse IgG1 not a human isotype, it is not even the closest mouse equivalent of human IgG1. Human IgG1 is not the equivalent of mouse IgG1 but rather of mouse IgG2a (see Paul *et al.*, *Fundamental Immunology*, 838, *supra*). Accordingly, Walker does not disclose or suggest an antibody having a human IgG1 isotype, as claimed. Therefore, combination of Nettleship with

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Walker does not lead to the claimed invention. Moreover, insofar as Walker provides any relevant teaching at all, by discussing a mouse antibody that does not have the equivalent isotype of human IgG1, Walker teaches away from the claimed invention.

Better discusses improved immunogenicity and half-life of chimeric antibodies relative to mouse antibodies. However, such improvements are attributed to the "human specific" properties of chimerics (Better at col. 3, lines 42-52) rather than to any particular human isotype. Better provides no indication that the human IgG1 isotype is better than any other human isotype for providing improved immunogenicity or half-life. Insofar as the rejection may intend to apply Nettleship with Better alone, Better provided no motivation to select a human IgG1 isotype rather than any other human isotype to combine with Nettleship. Thus, the combination of references would not have impelled the artisan to the specific combination claimed. Insofar as Better is cited merely as additional motivation to combine Nettleship with Walker, the alleged additional motivation does not change the fact that combination of Nettleship and Walker does not result in the claimed invention

Although it is unclear whether Friedland is meant to be included in the primary rejection, appellant notes that Friedland teaches away from the claimed invention. Friedland reports that the Fab form of an antibody stained similarly to an intact antibody and was advantageous relative to the intact antibody because of its rapid clearance. Because a Fab form of an antibody lacks most of a heavy chain constant region, and the heavy chain constant region determines the isotype of an antibody, this result would have suggested that the isotype of the antibody was irrelevant.

For these reasons, none of Walker, Better or Friedland, individually or in combination, would have provided any motivation to select the human IgG1 isotype thereby compensating for the acknowledged deficiency in Nettleship.

In attempting to rebut appellant's position, the Examiner points to col. 3, lines 43-45 of Better as specifically motivating selection of human IgG1 isotype. This passage reads as follows:

Unlike mouse antibodies, the human specific properties of the chimeric antibodies lower the likelihood of an immune response to the antibodies and result in prolonged survival in the circulation through reduced clearance. Moreover, using the methods of the invention, any desired antibody isotype can be combined with any particular antigen combining site.

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The first sentence merely states the undisputed fact that that chimeric antibodies have lower immunogenicity and clearance relative to mouse antibodies. The second sentence states that it is feasible to construct a chimeric antibody with any desired isotype but does not indicate why the human IgG1 isotype would be desirable over other isotypes in the context of the presently claimed methods. Feasibility without desirability is not enough for obviousness (*In re Fritsch, supra*). Because the above passage would not have provided any guidance of which human isotype would be desired in what circumstances, and particularly not in a method of treatment or prophylaxis of a disease characterized by amyloid deposits of A β , the passage would not have impelled the artisan to the specific combination claimed.

7.3.5 Lack of Reasonable Expectation of Success

The prior art cannot be modified or combined to reject claims as prima facie obvious without a reasonable expectation of success. *In re Merck & Co, Inc.*, 231 USPQ 375, 379 (Fed. Cir.1986). Here, it is respectfully submitted that the references, whether considered individually or in combination, did not provide a reasonable expectation of success. The only one of the cited references to discusses treatment of Alzheimer's disease in any detail, Nettleship, is entirely prophetic. Nettleship discusses assays by which he proposes to identify suitable compounds, but does not identify any suitable compounds, much less show they have any activity useful for treating Alzheimer's disease. Walker and Better are both directed to detection of amyloid deposits. Insofar as these references provide any information relevant to therapeutics, it is to emphasize the difficulty of delivering antibodies to the brain due to the blood brain barrier. For example, Friedland *et al.* state that they "do not anticipate passage of the labeled MAb through the blood-brain barrier" (p. 112, second column, first paragraph), and Walker states that the "blood-brain barrier prevents the passage of many types of molecules from the bloodstream to the brain . . . rendering vascular delivery of ligands to A β problematic" (at p. 377, first column, first paragraph). Better does not discuss treatment of Alzheimer's disease at all. The teachings of these references must be viewed in the context that before the priority date of the invention, Alzheimer's was regarded as an untreatable disease for which considerable effort had already been expended in fruitless search of a cure. In this context, it is respectfully submitted that individually and collectively the cited references did not provide a reasonable expectation of success of practicing the claimed methods.

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7.3.6 Dependent Claims

Claims 22, 23, 95 and 96 specify dosages of at least 1 mg/kg and 10 mg/kg body weight. The Examiner provides a conclusionary assertion that Nettleship appears to be enabling for determination of dosages (final office action at p. 8, third paragraph). The Examiner also takes the view that the claimed dosages are met by Friedland's disclosure of a dosage of 10 microgram per mouse based on a mouse weight of 10 g, corresponding to 1 mg/kg.

Although in some circumstances, the Examiner may be correct that it may be a routine matter to determine dosage and frequency of administration of a pharmaceutical, this is not the case when there are as many variables to explore as are left unresolved by Nettleship. Nettleship does not provide any exemplary compound for use in treatment. Nettleship also does not show that any particular end point can be achieved as an indication of successful treatment in a patient (for example, prevention of further deposition of A β in the brain, reduction of A β in the brain, reduction of toxicity to neuronal cells, or improvement in behavioral symptoms). In view of this lack of guidance, the practitioner is left to experiment with numerous variables simultaneously (agent, dosage, frequency, site of administration and end point). As the number of variables is increased, the number of permutations of variables increases exponentially. This results in a corresponding increase in the extent of experimentation required to find operable permutations. Moreover, this experimentation would have to have been performed in the context of a search for a first treatment of a hitherto untreatable disease for which considerable effort had already been expended in search of a cure. To have succeeded in devising actual compounds and a suitable regime for administration so as to prevent or treat Alzheimer's disease would have been considered far from being a routine matter based on the limited discussion provided by Nettleship. Moreover, if one were to attempt to supplement Nettleship's teaching based on Friedland, one would be lead away from the claim invention. The Examiner's estimate of 10 g as being the weight of a mouse is unduly low, the usual weight being about 50 g (see specification at p. 70, line 25). A dosage of 10 micrograms, as discussed by Friedland, to a mouse of 50 g actually corresponds to a dosage of 0.2 mg/kg. Therefore, insofar as the skilled person would rely on Friedland at all in selecting a dosage for detection of amyloid deposits, Friedland teaches away from the claimed invention.

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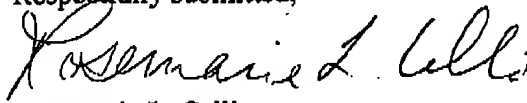
Claims 31 and 98 specify an antibody that binds to A β without binding to amyloid precursor protein. The Examiner has not identified any teaching in any of the cited references regarding such an antibody.

CONCLUSION

For these reasons, it is respectfully submitted that the rejection under 35 U.S.C. § 103 should be reversed and the application remanded to the Examiner for submission of terminal disclaimers to address the obviousness-type double patenting rejections.

Dated: June 27, 2006

Respectfully submitted,



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8. CLAIM APPENDIX

1. A method of treating a disease characterized by an amyloid deposit comprising A β peptide, the method comprising administering to a patient having the disease an antibody that specifically binds to A β peptide, in a regime effective to or treat the disease, wherein the antibody is a chimeric or humanized antibody, or a human monoclonal antibody and the antibody is of isotype human IgG1.

2. The method of claim 1, wherein the disease is Alzheimer's disease.

4. The method of claim 1, wherein the patient is a human.

6. The method of claim 1, wherein the patient is under 50.

7. The method of claim 1, wherein the patient has inherited risk factors indicating susceptibility to Alzheimer's disease.

8. The method of claim 1, wherein the patient has no known risk factors for Alzheimer's disease.

10. The method of claim 2, wherein the antibody is a human monoclonal antibody.

11. The method of claim 2, wherein the antibody is a humanized antibody.

12. The method of claim 2, wherein the antibody is a chimeric antibody.

17. The method of claim 1, further comprising administering an effective dosage of a second antibody that binds to the amyloid deposit or a component thereof.

21. The method of claim 2, wherein a chain of the antibody is fused to a heterologous polypeptide.

22. The method of claim 2, wherein the dosage of antibody is at least 1 mg/kg body weight of the patient.

23. The method of claim 2, wherein the dosage of antibody is at least 10 mg/kg body weight of the patient.

24. The method of claim 2, wherein the antibody is administered with a carrier as a pharmaceutical composition.

31. The method of claim 2, wherein the antibody specifically binds to A β peptide without binding to full-length amyloid precursor protein (APP).

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32. The method of claim 1, wherein the antibody is administered intraperitoneally, orally, subcutaneously, intramuscularly, topically or intravenously.

35. The method of claim 1, further comprising monitoring the patient for level of administered antibody in the blood of the patient.

36. The method of claim 1, wherein the antibody is administered in multiple dosages over a period of at least six months.

37. The method of claim 1, wherein the antibody is administered as a sustained release composition.

82. A method of prophylaxis of a disease characterized by an amyloid deposit comprising A β peptide, comprising administering to a patient an antibody that specifically binds to A β peptide, in a regime effective to effect prophylaxis of the disease, wherein the antibody is a chimeric or humanized antibody, or a human monoclonal antibody, and the antibody is of isotype human IgG1.

83. The method of claim 82, wherein the disease is Alzheimer's disease.

84. The method of claim 82, wherein the patient is a human.

85. The method of claim 82, wherein the patient is under 50.

86. The method of claim 82, wherein the patient has inherited risk factors indicating susceptibility to Alzheimer's disease.

87. The method of claim 82, wherein the patient has no known risk factors for Alzheimer's disease.

88. The method of claim 82, wherein the antibody is the human monoclonal antibody.

89. The method of claim 82, wherein the antibody is the humanized antibody.

90. The method of claim 82, wherein the antibody is the chimeric antibody.

93. The method of claim 82, further comprising administering an effective dosage of a second antibody that binds to the amyloid deposit or a component thereof.

94. The method of claim 82, wherein a chain of the antibody is fused to a heterologous polypeptide.

95. The method of claim 82, wherein the dosage of antibody is at least 1 mg/kg body weight of the patient.

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96. The method of claim 82, wherein the dosage of antibody is at least 10 mg/kg body weight of the patient.
97. The method of claim 82, wherein the antibody is administered with a carrier as a pharmaceutical composition.
98. The method of claim 82, wherein the antibody specifically binds to A β peptide without binding to full-length amyloid precursor protein (APP).
99. The method of claim 82, wherein the antibody is administered intraperitoneally, orally, subcutaneously, intramuscularly, topically or intravenously.
100. The method of claim 82, further comprising monitoring the patient for level of administered antibody in the blood of the patient.
101. The method of claim 82, wherein the antibody is administered in multiple dosages over a period of at least six months.
102. The method of claim 82, wherein the antibody is administered as a sustained release composition.

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9. **EVIDENCE APPENDIX**

Paul *ed.*, *Fundamental Immunology*, Raven Press (1993), p. 838, attached to response of August 14, 2005, entered with office action of November 29, 2005

WO 00/72880, IDS cite no. 322, filed October 15, 2002, entered with office action of October 16, 2003 at p. 18, lines 15-17.

Bard *et al.*, PNAS, 100:2023-2028 (2003) IDS April 28, 2005 cite no. 550, p. 2027, second column, third paragraph, entered into record with office action of November 29, 2005.

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10. RELATED PROCEEDINGS APPENDIX

An appeal is pending in a related case, U.S. Application No. 09/723,765. However, the claims and issues are significantly different. No Board or Court decisions have issued in the related case.

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